in the control hemispheres (intact side). Table 1 provides a comparison of the ratios of the cerebrovascular areas on the intact side and on the ischemic (lesion) side following the administrations of ginsenoside Rb_1, in doses of 6 μg/day and 60 μg/day. Data are represented as a mean ± SE. Statistical analyses were conducted by ANOVA + Fisher's PLSD. As indicated in Table 1, the ratios of the cerebrovascular areas showed no significant difference between the control side and the ischemic side. This indicated that, as a result of intravenous administration of ginsenoside Rb_1 within 28 days after permanent occlusion of the MCA, the cerebrocortical vascular networks in the ischemic penumbra have been almost completely regenerated and reconstructed. In addition, the ratio of the cerebrovascular area in the ischemic penumbra of the parietal lobe is quite naturally, significantly reduced as compared with that on the intact side immediately after permanent occlusion of the MCA.

Further, paraffin sections prepared from brain samples at the level 3.6 mm posterior to bregma were subjected to Nissl staining, and the ratio of the left thalamic area to the right thalamic area (ischemic side/intact side × 100) at the same level was measured. The groups administered with ginsenoside Rb_1 showed significantly higher values than the ischemic group administered with vehicle (physiological saline), and they exhibited the value close to that of the sham-operated group. These findings indicate that secondary atrophy of the thalamus

generated after cerebrocortical infarction is almost completely inhibited by intravenously administered ginsenoside Rb_1. In addition, we have investigated the histological patterns of the ventral posterior nucleus of the thalamus (VP thalamic nucleus) which has reciprocal synaptic connections (fiber connections) with the ischemic core of the cerebral cortex. Fig. 7A shows the thalamic VP nucleus of a sham-operated animal; Fig. 7B shows the thalamic VP nucleus of an ischemic animal administered with physiological saline; and Fig. 7C shows the thalamic VP nucleus of an ischemic animal administered with ginsenoside Rb_1 (60 μ g/day). Comparing the ischemic animal infused with physiological saline (Fig. 7B), with the ischemic animal intravenously infused with ginsenoside Rb_1 (Fig. 7C), reveals that a large number of nerve cells (neurons) survived significantly without the secondary degeneration in the ginsenoside Rb_1-treated VP thalamic nucleus. Consequently, it has been shown that intravenously administered ginsenoside Rb_1 inhibits the secondary degeneration of the thalamus.

We have further investigated the effects of intravenously administered ginsenoside Rb_1 on spinal cord injury, which is an intractable disease causing secondary degeneration of the nervous tissues. When a compression load is added to a spinal cord segment such as the lower thoracic cord, not only nerve cells in the gray matter of that segment but also fiber tracts

or pathways in the white matter are damaged. The original damage in the white matter also develops towards the distal region (caudal region) as well as causing secondary degeneration in the efferent neurons or origins of the fiber tracts or pathways damaged, namely the upper nerve cell bodies (i.e. efferent neurons or origins) which project nerve fibers to the fiber tracts or pathways damaged. Accordingly, the damage to the fiber tracts or pathways of the white matter in the lower thoracic cord, which results from the compression load, caused paraplegia of both hindlimbs. Thus, this paraplegia occurs due to the secondary degeneration of the efferent neurons or origins (nerve cell bodies) projecting to the primary lesion of the fiber tracts or pathways in the thoracic cord, and due to secondary degeneration of fiber tracts or pathways distal to the lower thoracic cord (i.e. lumbar and sacral cords). Further, the damage to the lower thoracic cord interrupts the innervation from the upper brain to the lumbar and the sacral cords. As a result, the secondary degeneration of nerve cells or neurons in the gray matter of the lumbar and sacral cords is aggravated to make paraplegia of both hindlimbs irreversible. We used Wistar rats, (weighing about 300 g), which were loaded with 20 g of compression to the lower thoracic cord for 20 minutes, as an animal model for spinal cord injuries.

The rats were anesthetized by inhalation of halothane in a mixture of nitrous oxide and oxygen and were loaded with 20